Pollution and People

Asbestos—can it be used safely?

DAPHNE GLOAG

Asbestos has been mined in small amounts on and off since the Stone Age,[1] having been used for diverse purposes such as making pottery, wicks, and shrouds. Industrially, however, it has been used for only a century. Its production has increased exponentially during this time,[2 3] and it is said to have some 3000 uses.[2] For some purposes, in fact, substitutes are available; and for insulation asbestos has largely been replaced in the last decade. But for many uses there is no equally satisfactory and economic alternative, and for friction materials it is held to be irreplaceable at present. Moreover, millions of tons are already present in our buildings and elsewhere. Thus a ban on all types of asbestos (see box) does not appear to be a present possibility, though some argue that it is.[4] And would such a ban be desirable —are there health hazards at low levels of exposure that outweigh the valuable and sometimes life-saving properties of the material?

As with so many pollutants, several scientific uncertainties remain. There is plenty of evidence that incidental as well as occupational exposure to asbestos may be hazardous; but is there a threshold for cancer, as there appears to be for asbestosis,[3] below which no harm can be expected? Without knowing more about dose-response relationships and the mechanisms of damage it is difficult to be sure how far the risk at low levels can be extrapolated, for the different fibre types, from occupational data. Nevertheless, the persistence of asbestos fibres in the tissues makes it at least theoretically plausible that cancer could on occasion be induced by extremely small quantities.

Though we have considerable data from epidemiological and case studies some provisos have to be made. Firstly, mesotheliomas in particular may have an extremely long latent period —commonly 20-50 years—so that some may be missed in follow-up studies. Secondly, it may be difficult to obtain a full history of exposure to asbestos, at work and elsewhere, covering a person's entire life[5 6]—and no less difficult to exclude such exposure. Some jobs also turn out to have given exposure to asbestos that comes to light only after considerable investigation.[7] Thus associations with the fibre may sometimes be missed, or a tumour may be wrongly attributed to some more casual exposure to asbestos.[1] Thirdly, the control groups used are not always appropriate[8]; and, fourthly, the dust concentrations to which patients have been exposed are often difficult or impossible to estimate. A further problem (see below) is that the types of asbestos associated with particular cases are not always obvious.

Types of cancer associated with asbestos

The main tumour associated with asbestos is lung cancer, though clearly it cannot be attributed to it with confidence in a given case if the patient has smoked and asbestos exposure has been low. The first epidemiological study, of a factory in Rochdale, showing the link beyond doubt was reported in 1955.[9] Mesotheliomas are less common but the relationship is stronger. They were previously thought an extreme rarity (though recognised in the nineteenth century) and were first found in considerable numbers, and associated with crocidolite, in the mining area of Cape Province, South Africa.[10] Apart from lung cancer and pleural and the rather less common peritoneal mesothelioma—found in workers heavily exposed to crocidolite—increases in gastrointestinal cancers and to a lesser extent cancer of the larynx have been reported in some studies.[1-3] Some gastrointestinal cancers, however, may be misdiagnosed peritoneal mesotheliomas. The importance of distinguishing between different types of asbestos, because of their different degrees of hazard, was not realised until the mid-1950s.

People had assumed that the asbestos regulations of 1931, which resulted from the studies of Dr E R A Merewether, had solved the asbestos problem, though cases would continue to occur for a time in workers who had experienced the old conditions. This was optimistic for several reasons. One was the increase in smoking: though lung cancer does occur in some non-smokers exposed to asbestos, smoking may multiply the risk from asbestos exposure many times[11-13] and thus would make the disease much more prominent than before in asbestos

Asbestos and its uses

Asbestos is the name given to several different silicates with a fibrous, crystalline structure,[1 2] widely distributed in the earth's crust and naturally present in dust in small amounts. There are two main types—amphibole and serpentine. The amphiboles used in industry are *crocidolite* ("blue asbestos"), no longer imported into Britain as it is the most dangerous; *amosite* ("brown asbestos"), whose use is now increasing most rapidly; and *anthophyllite* (now hardly used). The serpentine *chrysotile* ("white asbestos") is much the most common type in nature; its fibres have a curly structure making them less apt to penetrate airways and tissues, though they can split into fine fibrils. Because it confers heat resistance (and acid resistance in the case of crocidolite), insulation, and reinforcement and yet is a flexible material, asbestos is extensively used[3]—for example, for fire protection and heat-resistant materials, in cement for building and pipes, for electrical and other insulation, and for friction materials. The risks of asbestosis and cancer come from inhaling free asbestos fibres, and the raw and unbound material (for example, insulation products and sprayed asbestos) is therefore more hazardous than asbestos bound in, say, cement and plastic floor tiles.

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workers. Secondly, less dusty conditions made workers less likely to succumb to asbestosis or tuberculosis and more likely to survive long enough to develop cancers, especially mesotheliomas with their very long latent periods. Moreover, smaller exposures seem to be capable of inducing lung cancer and mesothelioma than asbestosis.[1] Thirdly, many more jobs entailed exposure to asbestos than were covered by the regulations; in some cases indeed workers were being only indirectly exposed, through working near dust-producing operations. Lastly, workers contaminated their relatives; and people living in the neighbourhood of mines and factories could also be at risk. The various sources of evidence have been summarised.[2]

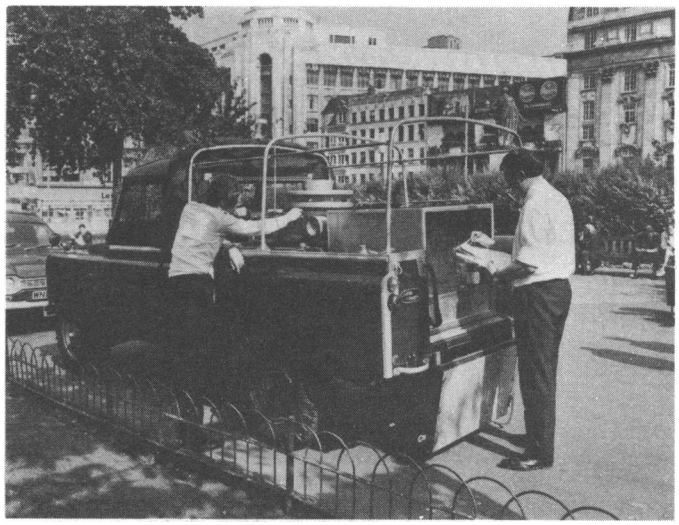
"Environmental" cancers?

The 1960 report from South Africa was the first evidence of the environmental hazard of crocidolite.[10] Some of the victims had merely lived near the mines or a mill, or worked at a clerical job there.[10] But exposure to the dust may have been considerable: children, for instance, played on asbestos dumps. A London study showed that an excessive proportion of patients had lived within half a mile (four-fifths of a kilometre) of a factory processing amphibole asbestos.[5] Some patients had been exposed to the dust through relatives who worked with asbestos and often appeared to have been heavily contaminated—for instance, by washing their work clothes; one said that her husband (a docker) had returned from work "white with asbestos" and that she had brushed him down every evening.[5] Studies including a category of "possible exposure" have included patients who had briefly engaged in, say, "do-it-yourself" jobs handling asbestos.[7] But with these, and with possible "neighbourhood cases," how likely is a causal link?

Though mainly an asbestos cancer, mesothelioma is thought to be "spontaneous" in a proportion of cases.[6] In a particularly thorough study the annual rate in Canada in 1972 was estimated as 2·8 per million in men and 0·7 in women—the latter presumably nearer the "natural" background rate.[14] Interviews to ascertain occupational history and possible exposure to asbestos were conducted "blind," with the interviewers ignorant of which were cases and which controls to avoid bias in the questioning. Occupational exposure to asbestos was found in nearly half the cases in Canada and about two-thirds in the United States; the overall proportion in women, however, was only 5%. This, of course, begs the question of whether any given case with no occupational history might have been caused by some undiscovered "casual" contact with asbestos. A lung fibre study found 87 out of 100 cases of mesothelioma to have high fibre counts.[6] Brief exposure might be harmful, it has been suggested, if sufficiently intense[6]; it is sometimes claimed that intermittent exposure may be more dangerous than continuous, given the same cumulative doses, because it could be the peak dust levels that are responsible.[1 8] There is no information, however, about actual dust concentrations to confirm such a view. In reported cases where mesotheliomas have been associated with only a day working with asbestos (see IARC,[2] table 22) there are other possibilities, notably "spontaneous" tumours and more prolonged occupation that has been missed. On the other hand, the fibre study just mentioned found extremely high fibre counts in the lungs of some who had been exposed in their jobs for only three months.[6] Rather than speculate, however, we may usefully look at information on the relative risks of the different types of asbestos and such dose-response data as may be gleaned from occupational studies.

Fibre types and dose-response relationships

There is a generally accepted gradient of risk, for both lung cancer and mesothelioma, for the three main types of asbestos, from chrysotile through amosite to crocidolite[1 15]; this is reflected in the current industrial limits.[16] But use of a single fibre type is rare in industry,[17] and different jobs give different risks (insulation work, using dry asbestos, having been particularly hazardous[15]); so the matter is complex.



Sampling asbestos in the atmosphere, Piccadilly, Manchester. Photograph by courtesy of the Asbestos Information Centre.

Most uncertainty has surrounded chrysotile as few factory populations have been exposed to this fibre in isolation. The largest series of workers exposed to chrysotile alone consists of over 11 000 chrysotile miners and millers in Quebec Province; this showed an increase in total mortality and particularly in deaths from lung cancer and asbestosis—but only in those exposed to unacceptable dust concentrations by present standards.[13] Only 11 pleural mesotheliomas occurred in the 50 years. A paper by the same workers[15] reviewing the data concludes that for those engaged in chrysotile production the risk of mesothelioma is perhaps three to six times greater than in the general population, compared with a risk 100-200 times greater in insulation workers, exposed to chrysotile but also crocidolite or amosite.[15] Whether this means that chrysotile has more effect in these conditions or that exposure to the amphiboles was responsible or that these workers were exposed to a cocarcinogen is not clear.[15]

An American investigation was based on one of the few factories that processes almost exclusively chrysotile, which had for long had relatively low dust levels and which possessed good data on the exposure of its textile workers.[18] This showed for lung cancer a standardised mortality ratio of 223 for the lowest cumulative doses. Only one of the 191 deaths was due to mesothelioma. In a cohort of workers using mainly chrysotile there were 10 mesotheliomas, an incidence of 0·05%; but a statistical analysis suggested that for a man employed continuously the life-long risk could have been up to 10%.[19] Some crocidolite is said to have been used in this factory, however, during two appreciable periods. At a factory manufacturing friction materials, where there were also 10 deaths from pleural mesothelioma, all but two of the cases had definitely been exposed to crocidolite even though chrysotile was mainly used, the association with crocidolite having a probability of 0·06.[20] A significant excess of cancer of the lung and pleura occurred in those employed before 1942, when conditions were dustier. The consensus of the various findings is that crocidolite is considerably more carcinogenic than chrysotile. Exposure to amosite alone is rare but the experience of American insulation workers suggests that it is more hazardous than chrysotile[21]; these workers could, however, have been exposed to some crocidolite.

Some reports present enough data to suggest that the risk of cancers is proportional to the accumulated dose of fibre, or at least are not inconsistent with a linear dose-response relationship.[1 3] For lung cancer in chrysotile miners and millers the

Canadian results show a clearly linear relationship[13]; and in the American study risk was also in proportion to exposure.[18] Mesotheliomas have been too few for inferences about dose-response curves.

For crocidolite and for mixtures of fibres the risks of both lung cancer and mesothelioma appear to be roughly proportionate to the likely dose of fibre.[3 22 23] In the amosite workers mentioned above there were increased risks of lung cancer, all cancers, and all asbestos diseases that were related to duration and intensity of exposure, men employed for only a month showing some excess risk.[21]

The EEC report concludes that there is "suggestive evidence" from the epidemiological studies for a threshold limit for asbestos exposure below which excess cancer risk is small or non-existent, but that no adequate data to establish such a limit are available.[1] The report adds that for mesothelioma induction the relevant exposure may be impossible to estimate since the peak dust levels are probably important here. However that may be, for chrysotile at low levels the epidemiological data are reassuring: even if, as with many other carcinogens, there is no threshold the diminishing probability of cancer induction must result in exceedingly small risks. In the next article, after looking at some fibre studies, I will discuss the environmental aspects of exposure to asbestos.

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References

1 Zielhuis RL. *Public health risks of exposure to asbestos.* Report of a working group of experts prepared for the Commission of the European Communities, Directorate-General for Social Affairs, Health and Safety Directorate. Oxford: Pergamon Press, 1977.

2 International Agency for Research on Cancer. *Asbestos.* IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol 14. Lyon: IARC, 1977.

3 Acheson ED, Gardner MJ. The ill-effects of asbestos on health. In: Health and Safety Commission. *Asbestos. Final report of the advisory committee.* Vol 2: Papers prepared for the advisory committee. London: HMSO, 1979:7-83.

4 Dalton A. *Asbestos—killer dust.* London: British Society for Social Responsibility in Science, 1979.

5 Newhouse ML, Thompson H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Industr Med* 1965;22:261-6.

6 Whitwell F, Scott J, Grimshaw M. Relationship between occupations and asbestos-fibre content of the lungs in patients with pleural mesothelioma, lung cancer, and other diseases. *Thorax* 1977;**32**:377-86.

7 Lieben J, Pistawka H. Mesothelioma and asbestos exposure. *Arch Environ Health* 1967;**14**:559-66.

8 Enterline PE. Pitfalls in epidemiological research. An examination of the asbestos literature. *Ann Occup Health* 1976;**18**:150-6.

9 Doll R. Mortality from lung cancer in asbestos workers. *Br J Industr Med* 1955;**12**:81-6.

10 Wagner JC, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in the north western Cape Province. *Br J Industr Med* 1960;**17**:260-71.

11 Selikoff IJ. Asbestos exposure, smoking, and neoplasia. *JAMA* 1968; **204**: 106-12.

12 Berry G, Newhouse ML, Turok M. Combined effect of asbestos exposure to smoking on mortality from lung cancer in factory workers. *Lancet* 1972;ii:476-9.

13 McDonald JC, Liddell FDK, Gibbs GW, Eyssen GE, McDonald AD. Dust exposure and mortality in chrysotile mining, 1910-75. *Br J Indutsr Med* 1980;**37**:11-24.

14 McDonald AD, McDonald JC. Malignant mesothelioma in North America. *Cancer* 1980;**46**:1650-6.

15 McDonald JC, McDonald AD. Epidemiology of mesothelioma from estimated incidence. *Prev Med* 1977;**6**:426-46.

16 Health and Safety Commission. *Asbestos.* Vol 1. *Final report of the Advisory Committee on Asbestos.* London: HMSO, 1979.

17 Pooley FD. An examination of the fibrous mineral content of asbestos lung tissue from the Canadian chrysotile mining industry. *Environmental Research* 1976;**12**:281-98.

18 Dement JM, *et al.* Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. In: Walton H, ed. *Proceedings of fifth international symposium on inhaled particles* (British Occupational Hygiene Society), Cardiff, 1980. Oxford: Pergamon Press (in press).

19 Peto J. The hygiene standard for chrysotile asbestos. *Lancet* 1978;ii:484-90.

20 Newhouse ML, Berry G, Skidmore JW. A mortality study of workers manufacturing friction materials with chrysotile asbestos. In: Walton H, ed. *Proceedings of fifth international symposium on inhaled particles* (British Occupational Hygiene Society), Cardiff, 1980. Oxford: Pergamon Press (in press).

21 Seidman H, Selikoff IJ, Hammond EC. Short-term asbestos work exposure and long-term observation. *Ann N Y Acad Sci* 1979;**330**:61-89.

22 Newhouse ML, Berry G. Pattern of mortality in asbestos factory workers in London. *Ann N Y Acad Sci* 1979;**330**:53-60.

23 Newhouse ML, Berry G. Predictions of mortality from mesothelial tumours in asbestos factory workers. *Br J Industr Med* 1976;**33**:147-51.

What is the best form of treatment for a varicose ulcer?

The principle of treating a varicose ulcer is simplicity itself; it is to reverse the high venous pressure in the affected leg. If the patient is put to bed with the leg raised rapid healing will take place, but this is usually not practicable. Ambulant treatment therefore comprises firm continuous elastic compression of the leg, but this can be combined with keeping the leg raised at night in bed or when sitting down. Standing is forbidden, but walking about while under supportive treatment is encouraged, since this favours venous return. The ulcer itself requires no more than gentle cleaning with saline and protection with a simple sterile gauze dressing—no local antibiotics, no steroids, no ointments, no creams, and no lotions. There is no evidence that these help in any way and in too many cases merely produce skin sensitivity. The simplest technique is to use Elastoplast bandaging, but because sensitivity will occur to this in a very high proportion of patients the skin of the leg must be protected from direct contact with the Elastoplast. The ulcer is therefore covered with a simple sterile dressing, the leg then bandaged with viscopaste from the base of the toes to below the knee (zinc paste and coaltar bandage (Coltapaste) may be used when the skin is dry and scaly or zinc paste and ichthamanol bandage (Ichthopaste) when the skin is moist and eczematous). Over this protective layer the Elastoplast bandage is applied from the metatarsophalangeal joints to below the knee, from below upwards, carefully avoiding kinks and constrictions. Normally the bandage may be left in place for two to three weeks, although the maximum is about six weeks. If there is a heavy discharge the bandaging may initially require weekly change. Associated varicose veins may be treated in most cases by injection technique[1] or in some cases, with gross varices extending to the groin, flush ligation of the vein at the saphenofemoral junction at the groin may be necessary. Once the ulcer has healed, it is essential that the patient goes on supporting the leg by a full length elastic stocking worn during the day and is encouraged to continue active exercises and keep the leg raised when resting.

1 Fegan G. *Varicose veins, injection sclerotherapy* London: Heinemann Medical, 1967.

Are women with Kartagener's syndrome or any of the other immotile cilia syndromes infertile?

There has been at least one pregnancy recorded in a woman with immotile cilia syndrome. In men affected by Kartagener's syndrome and other types of immotile cilia syndrome infertility is the rule, but it appears that women may have children.